Understanding the Role of Longitudinal Arterial Wall Motion in Blood Circulation from the Perspective of a Piano String *

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***Abstract*— This work is aimed to establish engineering theories of the coupled longitudinal and radial motion of the arterial wall. By treating the arterial wall as a piano string in the longitudinal direction and as a viscoelastic material in the circumferential direction, and considering pulsatile pressure and wall shear stress from axial blood flow in an artery, the fully-formed governing equations of the coupled motion of the arterial wall are obtained and are related to the engineering theories of axial blood flow for a unified engineering understanding of blood circulation in the cardiovascular (CV) system. The longitudinal wall motion and the radial wall motion are essentially a longitudinal elastic wave and a transverse elastic wave, respectively, traveling along the arterial tree, with their own propagation velocities dictated by the physical properties and geometrical parameters of the arterial wall. The longitudinal initial tension is essential for generating a transverse elastic wave in the arterial wall to accompany the pulsatile pressure wave in axial blood flow. Under aging and subclinical atherosclerosis, propagation of the two elastic waves and coupling of the two elastic waves weakens and consequently might undermine blood circulation.**

***Clinical Relevance*— Essential role of the longitudinal wall motion in achieving the radial wall motion and thus blood circulation is identified and will be useful for interpreting the measured coupled arterial wall motion for clinical values.**

I. Introduction

The essential function of the cardiovascular (CV) system is blood circulation throughout the body. Instead of axial blood flow in an artery, the arterial wall takes >90% energy output of the left ventricle, and thus the arterial wall motion is critical for achieving blood circulation [1]. Atherosclerosis has been identified as the dominant cause of CV diseases [2]. These may explain the reason why arterial indices are utilized for detection and diagnosis of CV diseases. For instance, arterial stiffness, mostly in terms of pulse wave velocity (PWV), is indicative of arterial elasticity, and peripheral vascular resistance (PVR) is dictated by arterial radius at diastolic blood pressure [3].

Upon blood ejection from the left ventricle, the arterial wall undergoes radial motion and longitudinal motion simultaneously [4]. With neglect of the longitudinal wall motion, engineering theories have been well established for assessing arterial indices from measured arterial pulse signals and are widely used in clinical studies [2]. Generally speaking, these theories fall into two categories [2]: 1) a transverse elastic wave model, and 2) a viscoelastic material model. Built on the governing equations of axial blood flow with the radial wall motion as the boundary condition, the pulsatile pressure in an artery was found to be a propagation wave traveling along the arterial tree with the propagation velocity, PWV. Then, the radial wall motion was also considered as a transverse elastic wave accompanying the pulsatile pressure wave. Global/regional PWV are obtained by simultaneously measuring pulse signals at two artery sites (e.g., carotid versus femoral). The arterial wall is also treated as a viscoelastic material in the circumferential direction, which relates the pulsatile pressure to the radial wall motion at an artery site for assessing local arterial elasticity and viscosity [3].

In recent years, the longitudinal wall motion has been found to serve as a more sensitive and possibly earlier measure of subclinical atherosclerosis, as compared with those radial-based arterial indices [4]. Correlations between radial-based arterial indices and amplitudes of the longitudinal wall motion have also been identified [4], possibly implying coupling of the arterial wall motion between the two directions. By treating the arterial wall as an isotropic viscoelastic Koiter shell, Bukač and S. Čanić [5] derived the governing equations of the coupled longitudinal and radial motion of the arterial wall. However, these equations do not consider the inherent anisotropic nature of the arterial wall and lack conceptual clarity for revealing CV physiology. As such, engineering theories of the coupled wall motion are still unclear, making the role of the longitudinal wall motion in blood circulation unattainable.

This paper is aimed to establish engineering theories of the coupled longitudinal and radial motion of the arterial wall for their clinical applications. The author previously derived the governing equations of the coupled longitudinal and radial motion of the arterial wall, with its longitudinal initial tension being neglected [6]. Yet, the arterial wall is longitudinally pre-stretched and thus its longitudinal initial tension is expected to play a role in the arterial wall motion. Similar to the arterial wall, a piano string is pre-stretched and undergoes the coupled longitudinal and transverse motion [7]. In this paper, the arterial wall is treated as a piano string in the longitudinal direction and as a viscoelastic material in the circumferential direction, the contribution of axial blood flow to the arterial wall motion is considered as pulsatile pressure and wall shear stress. Consequently, the fully-formed governing equations of the coupled motion of the arterial wall are obtained and are further related to the governing equations of axial blood flow for a unified engineering understanding of blood circulation in the CV system. The established engineering theories of the coupled wall motion shed important insights on the CV physiology.

*This work is partially supported by National Science Foundation under Grant 1936005.

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II. Fully-Formed Governing Equations of the Coupled Motion of the Arterial Wall

A. The Arterial Wall in the Longitudinal Direction: a Piano String

As shown in Fig. 1(a), the arterial wall is subjected to diastolic blood pressure (DBP) and pulsatile pressure, Δp, in the radial direction. Blood flow rate, Q, along the longitudinal direction (x-axis) causes a wall shear stress, τ_w, acting on the arterial wall. The primary geometrical parameters of the arterial wall are its radius, r_0, at DBP and thickness, h, as shown in Fig. 1(b).

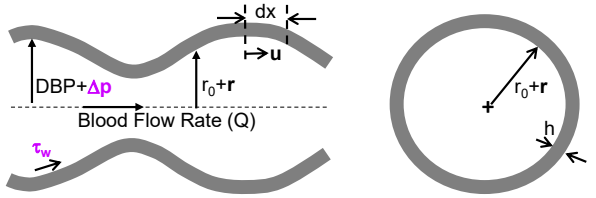

Figure 1. Schematics of the arterial wall (a) along the longitudinal direction (x-axis) with blood pressure $DBP+\Delta p$ in the radial direction and blood flow rate Q in the x-axis and undergoing radial motion $r(x, t)$ and longitudinal motion $u(x, t)$, (b) cross-section with its geometrical parameters

The arterial wall contains three layers (i.e., intima, media and adventitia) in the radial direction and is commonly treated as a thin-layered structure in clinical studies, due to a high ratio of r_0/h (e.g., 7~10 at the common carotid artery). Thus, the motion of a single point on intima-media complex is measured in clinical studies to represent the collective motion of the arterial wall and derive arterial indices. The arterial wall is inherently pre-stretched along the longitudinal direction [6], due to its layered structure. Thus, the arterial wall in the longitudinal direction (x-axis) is treated as a piano string with initial tension, T_0, and elasticity, E_{xx}. At the start/end of a pulse cycle, the arterial wall is under T_0 and in its equilibrium state: $u=0$ and $r=0$.

Fig. 2 shows an element dx of the arterial wall at the position, x, from the left ventricle. The displacements of the element in the x-axis and in the r-axis (or radial direction) are u and r, respectively. In a pulse cycle, the element dx is deformed into a space curve ds:

$$ds = \sqrt{(1+u_x^2)^2 + r_x^2}\cdot dx \tag{1}$$

where subscript x denotes the first-order derivative of a parameter with respect to x.

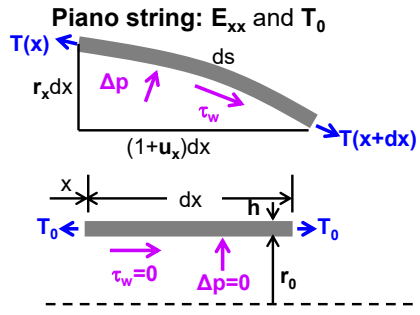

Figure 2. Schematics of the arterial wall element dx, its geometrical parameters, displacements, internal forces (in blue font), and external forces (in purple font).

By assuming small displacements in the two directions, the displacement gradients will be very small, and thus Eq. (1) can be simplified as:

$$ds = (1+u_x+\tfrac{1}{2}r_x^2)\cdot dx \tag{2}$$

In a pulse cycle, the tension in the arterial wall at the point that is originally at x is given by:

$$T = T_0 + E_{xx}S\cdot\frac{ds-dx}{dx} = T_0 + E_{xx}S\cdot(u_x+\tfrac{1}{2}r_x^2) \tag{3}$$

Note that E_{xx} is longitudinal elasticity on top of T_0, and $S = 2\pi r_0\cdot h$ is the cross-sectional area of the arterial wall.

The x-component, $T_{x\text{-}axis}$, and r-component, $T_{r\text{-}axis}$, of the tension force, T, become:

$$T_{x-axis} = T(1+u_x)\cdot dx/ds \tag{4a}$$

$$T_{r-axis} = T\cdot r_x\cdot dx/ds \tag{4b}$$

According to Eq. (2), the following relation holds:

$$dx/ds = 1-u_x-\tfrac{1}{2}r_x^2 \tag{5}$$

By substituting Eq. (5) into Eq. (4) and getting rid of the higher-order terms, Eq. (4) is simplified as:

$$T_{x-axis} = T_0(1-\tfrac{1}{2}r_x^2) + E_{xx}S\cdot(u_x+\tfrac{1}{2}r_x^2) \tag{6a}$$

$$T_{r-axis} = T_0\cdot r_x + (E_{xx}S-T_0)\cdot r_x\cdot(u_x+\tfrac{1}{2}r_x^2) \tag{6b}$$

Note that T_0 plays a role in the wall motion in the two directions, due to the radial displacement gradient, r_x.

B. Fully-formed Governing Equation of the Longitudinal Motion

Equating the net longitudinal force, $\partial T_{x-axis}/\partial x\cdot dx$, on the element dx to its inertial force in the x-axis gives rise to:

$$mu_{tt} = \partial T_{x-axis}/\partial x \tag{7}$$

where the subscript tt denotes the second-order derivative of a parameter with respect to time and $m = \rho_w 2\pi r_0 h$ is the mass of the arterial wall per unit length, with ρ_w denoting the wall density. Then, substituting Eq. (6a) into Eq. (7) and neglecting the higher-order terms gives rise to:

$$u_{tt} = c_l^2\cdot u_{xx} + \tfrac{1}{2}(c_l^2-c_0^2)\cdot(r_x^2)_x \tag{8}$$

where subscript xx denotes the second-order derivative of a parameter with respect to x, and c_0 and c_l are defined as

$$c_0 = \sqrt{\frac{T_0}{\rho_w 2\pi r_0 h}};\quad c_l = \sqrt{\frac{E_{xx}}{\rho_w}} \tag{9}$$

Based on Eq. (8), the longitudinal wall motion represents a longitudinal elastic wave, $u(x, t)$, with propagation velocity, c_l.

Axial blood flow contributes to the motion of the element dx through the wall shear stress, τ_w, and pulsatile pressure, Δp. The forces resulting from τ_w and Δp are:

$$F_{\tau_w} = \tau_w \cdot 2\pi r_0 dx \tag{10a}$$

$$F_{\Delta p} = \Delta p \cdot 2\pi r_0 dx \tag{10b}$$

By including the *x*-components of the above two forces into Eq. (8), the *fully-formed governing equation of the longitudinal wall motion* is obtained:

$$u_{tt} = c_l^2 \cdot u_{xx} + \tfrac{1}{2}(c_l^2 - c_0^2) \cdot (r_x^2)_x + \frac{\tau_w}{\rho_w h} + \frac{\Delta p}{\rho_w h} \cdot r_x \tag{11}$$

C. The Arterial Wall in the Circumferential Direction: a Viscoelastic Material

In the circumferential direction, the arterial wall is treated as a viscoelastic material in a circular ring shape with elasticity, $E_{\theta\theta}$, and viscosity, $\eta_{\theta\theta}$. Fig. 3 shows an element of $d\theta$ and unit length of the arterial wall. Its circumferential strain and stress are:

$$\varepsilon_{\theta\theta} = \mathrm{r} / \mathrm{r}_0 \tag{12a}$$

$$\sigma_{\theta\theta} = \mathrm{E}_{\theta\theta} \frac{r}{r_0} + \eta_{\theta\theta} \frac{r_t}{r_0} \tag{12b}$$

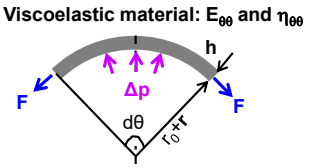

Figure 3. Schematic of an element of $d\theta$ and unit length of the arterial wall in the circumferential direction.

The sum of the *r*-component of the force, $F=\sigma_{\theta\theta}h$, and the force resulting from Δp should be equal to inertial force of the element in the *r*-axis [6]:

$$r_{tt} + \frac{\eta_{\theta\theta}}{\rho_w \mathrm{r}_0^2} r_t + \frac{\mathrm{E}_{\theta\theta}}{\rho_w \mathrm{r}_0^2} r = \frac{\Delta p \cdot r_0}{\rho_w h} \tag{13}$$

Neglecting the inertial term in Eq. (13) gives rise to the viscoelastic material model for assessing local arterial elasticity and viscosity. Eq. (13) indicates that the radial wall motion is transverse vibration at an artery site (at a fixed *x* position). Based on Eq. (13), the radial wall motion cannot propagate along the arterial tree (*x*-axis).

D. Fully-formed Governing Equation of the Radial Motion

Based on Fig. 2, equating the net radial force, $\partial T_{r-axis} / \partial x \cdot dx$, on the element *dx* to its inertial force in the *r*-axis gives rise to:

$$my_{tt} = \partial T_{r-axis} / \partial x \tag{14}$$

By substituting Eq. (6b) into Eq. (14), neglecting the higher-order terms, including the *r*-component of τ_w, Δp, and $F=\sigma_{\theta\theta}h$, the *fully-formed governing equation of the radial wall motion* becomes:

$$\begin{aligned} &r_{tt} + \frac{\eta_{\theta\theta}}{\rho_w \mathrm{r}_0^2} r_t + \frac{E_{\theta\theta}}{\rho_w \mathrm{r}_0^2} r = c_0^2 \cdot r_{xx} \\ &+ (c_l^2 - c_0^2) \cdot \left\{ r_x \cdot (u_x + \frac{1}{2} r_x^2) \right\}_x + \frac{\Delta p}{\rho_w h} - \frac{\tau_w}{\rho_w h} \cdot r_x \end{aligned} \tag{15}$$

Eq. (15) indicates that the radial wall motion is a transverse elastic wave, *r(x, t)*, with a propagation velocity of c_0. The second term on the right side of Eq. (15) represents coupling of *u(x, t)* to *r(x, t)*, but is a very small quantity (*x*-derivative of the multiplication of the two small qualities: u_x and r_x,). Thus, Eq. (15) can be reduced to:

$$r_{tt} + \frac{\eta_{\theta\theta}}{\rho_w \mathrm{r}_0^2} r_t + \frac{E_{\theta\theta}}{\rho_w \mathrm{r}_0^2} r = c_0^2 \cdot r_{xx} + \frac{\Delta p}{\rho_w h} - \frac{\tau_w}{\rho_w h} \cdot r_x \tag{16}$$

III. Discussion

A. Essential Role of the Longitudinal Motion in the Radial Motion of the Arterial Wall

Based on Eq. (11), the longitudinal wall motion represents a longitudinal elastic wave, *u(x, t)*, with the propagation velocity, c_l. Since the arterial wall is tethered to its surrounding tissues, τ_w and Δp serve as external sources to sustain *u(x, t)* traveling down the arterial tree. Although r_x is a small quantity, Δp (a few kPa) is much higher than τ_w (a few Pa) [8]. Therefore, the contributions of τ_w and Δp to *u(x, t)* might be comparable. A large difference between c_l and c_0 indicates high coupling of *r(x, t)* to *u(x, t)*.

Eq. (16) shows that the radial wall motion represents a transverse elastic wave, *r(x, t)*, with the propagation velocity, c_0. Both τ_w and Δp serve as external sources to sustain *r(x, t)* along the arterial tree. Since r_x is a small quantity, and τ_w is much smaller than Δp, Δp is the dominant source. Note that Δp is also needed to balance the circumferential stress, $\sigma_{\theta\theta}$, in the arterial wall. This may explain that the motion amplitudes in the two directions are comparable [9], although Δp is much higher than τ_w. Since T_0 dictates the propagation velocity of *r(x, t)* in the arterial wall, coupling of T_0 to *r(x, t)* is profound for moving the radial wall motion (transverse vibration) at an artery site down the arterial tree.

B. Connection between the Coupled Wall Motion and the Governing Equations of Axial Blood Flow

By neglecting the time-dependent terms in Eq. (13), the purely elastic model of the arterial wall in the circumferential direction is obtained [6]:

$$E_{\theta\theta} \cdot h \cdot r / r_0 = r_0 \cdot \Delta p \tag{17}$$

By adding Eq. (17) as the boundary condition to the governing equations (i.e., Navier-Stokes equation and continuity equation) of axial blood flow and neglecting their non-linear terms, the governing equation of pulsatile pressure can be obtained [6]:

$$\Delta p_{tt} = PWV^2 \cdot \Delta p_{xx} \tag{18}$$

with *PWV* being defined as [2, 6]:

$$PWV = \sqrt{\frac{E_{\theta\theta} h}{2 r_0 \rho_b}} \tag{19}$$

where ρ_b is blood density. Eq. (18) represents the well-established engineering essence for blood circulation: a pulsatile pressure wave, $\Delta p(x, t)$, in axial blood flow travels along the arterial tree with PWV.

As boundary condition, the radial wall motion should accompany $\Delta p(x, t)$ traveling along the arterial tree. According to Eq. (16), the radial wall motion is a transverse

elastic wave, $r(x, t)$, with its propagation velocity, c_0, which is dictated by T_0 in the longitudinal direction. Note that in the derivation of Eq. (18), $u(x,t)$ is neglected. In reality, there are three waves traveling along the arterial tree with their own velocity: $\Delta p(x, t)$ with PWV, $r(x,t)$ with c_0, and $u(x,t)$ with c_l.

C. Physiological Implications

Based on Eq. (13), the radial wall motion is transverse vibration and its natural frequency is given by [3]:

$$\omega_0 = \sqrt{\frac{E_{\theta\theta}}{\rho_w r_0^2}} \tag{20}$$

By neglecting the circumferential terms in Eq. (16), the radial wall motion is analogous to transverse vibration of a string with its initial tension, T_0. Then, the natural frequency of the arterial wall of length, L, becomes [7]:

$$\omega_0 = \frac{\pi}{L}\sqrt{\frac{T_0}{\rho_w 2\pi r_0 h}} \tag{21}$$

Smaller body size implies smaller arterial radius and shorter length of the arterial tree. Eq. (20) and (21) might explain the inverse relation of heart rate to animal body size.

From the ascending aorta to periphery, T_0, E_{xx}, and $E_{\theta\theta}$ increase [2]. Concomitant increase in c_0, c_l, and PWV might indicate the coordinated propagation of the three waves. T_0 is significant at young age and drops with aging [6]. Under aging and subclinical atherosclerosis, E_{xx} and $E_{\theta\theta}$ were found to decrease and increase, respectively, at the carotid artery [4]. Since reduction in T_0 was found to cause a larger decrease in E_{xx} than an increase in $E_{\theta\theta}$ [10], it might be inferred that aging and subclinical atherosclerosis cause reduction in both c_0 and c_l and a reduced difference between c_0 and c_l - a reduced coupling between $u(x, t)$ and $r(x, t)$, possibly undermining the propagation of the two elastic waves and thus blood circulation. As a result, PWV needs to increase to compensate for reduction in c_0 and c_l and reduced coupling between $u(x, t)$ and $r(x, t)$.

D. Energy Transport in the Longitudinal and the Radial Directions along the Arterial Tree

Here, energy transport associated with $u(x,t)$ and $r(x,t)$ is analyzed. The potential energy density (per unit mass) of the arterial wall in the x-axis and the r-axis is given by:

$$PE_{x-axis} = \int_0^u \left[c_l^2 \cdot u_{xx} + \tfrac{1}{2}(c_l^2 - c_0^2)\cdot (r_x^2)_x \right] \cdot du \tag{22a}$$

$$PE_{r-axis} = \int_0^r \left[c_0^2 \cdot r_{xx} + \frac{\eta_{\theta\theta}}{\rho_w r_0^2} r_t + \frac{E_{\theta\theta}}{\rho_w r_0^2} r \right] \cdot dr \tag{22b}$$

A large difference between c_0 and c_l indicates higher energy transfer from $r(x,t)$ to $u(x,t)$. As compared with the potential energy in $u(x, t)$, the potential energy in $r(x, t)$ is dominant, given that the arterial wall stores a large amount of potential energy in the circumferential direction. Thus, energy transport in the arterial tree is mainly carried by $r(x, t)$. Although the potential energy in $u(x, t)$ is low, $u(x, t)$ is essential to move $r(x, t)$ down the arterial tree.

As energy supply to $u(x, t)$ and $r(x, t)$ for sustaining their propagation, the work done by τ_w and Δp in the x-axis and the r-axis is:

$$\Delta W_{x-axis} = \frac{\tau_w}{\rho_w h}\cdot du + \frac{\Delta p}{\rho_w h}\cdot r_x \cdot du \tag{23a}$$

$$\Delta W_{r-axis} = \frac{\Delta p}{\rho_w h}\cdot dr - \frac{\tau_w}{\rho_w h}\cdot r_x \cdot dr \tag{23b}$$

Since the amplitudes of $u(x, t)$ and $r(x, t)$ are comparable [9], energy supply to $u(x, t)$ is much lower than that to $r(x, t)$, which is consistent with the difference in potential energy of the two elastic waves.

IV. Conclusion

In this paper, the fully-formed governing equations of the coupled longitudinal and radial motion of the arterial wall are derived and are related to the governing equations of axial blood flow for a unified engineering understanding of blood circulation in the CV system. For the first time, an essential role of the longitudinal initial tension of the arterial wall in transverse elastic wave propagation and thus blood circulation is identified. The established engineering theories of the coupled wall motion elucidate the effect of the physical properties and geometries of the arterial wall on blood circulation and will be useful in interpreting the measured coupled wall motion for their clinical values. Adding the influence of various arterial pathologies on the arterial wall (e.g., nonlinear behavior [11]) to the established engineering theories might contribute to a better understanding of their origin and progress on a mechanistic level in the future.

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